between the greater curvature of the stomach and the colon for a distance of approximately 12 cm. This appeared to fix the stomach firmly to the now nonyielding transverse colon. All three plications were done in a similar manner. Two interrupted No. 00 cotton sutures on either end reinforced a continuous row of No. 00 chromic catgut. All sutures were seromuscular. No gastroenterostomy was performed. At the completion of the operation, when the omentum was replaced in the peritoneal cavity it was noted that the stomach was firmly fixed in position. The patient tolerated the procedure very well. Gastric suction and parenteral alimentation were carried out for 48 hours. The patient was discharged from the hospital five days following the operation.

A month after the operation a gastrointestinal series and barium enema study were done (Figure 3). While the stomach showed normal mobility and pliability, there was not a vestige of the former hypermobility. Films taken in all positions, including deep Trendelenberg, demonstrated excellent fixation of the transverse colon and stomach.

Completely asymptomatic since the operation, she no longer feels any discomfort even when she assumes positions that formerly caused distress.

SUMMARY

1. A rare case of idiopathic gastric volvulus of the organo-axialis type is presented.
2. The literature on this subject is reviewed.
3. A new surgical method, used with excellent result, is presented for the treatment of this unusual condition.

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Calcified Meconium Abscess Causing Intestinal Obstruction in an Infant

Report of a Case and Review of the Subject

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THE PATIENT, a boy, was delivered by cesarean section on September 12, 1949, a full term, viable infant weighing 6 pounds 5 ounces.

The prenatal course was possibly of some significance in that in an automobile accident on June 21, 1949, the mother was thrown beneath the dashboard and struck her abdomen. The following day, although she complained of pain and tenderness in the midabdomen, there was no objective evidence of significant trauma, and the symptoms gradually subsided. On August 22, 1949, two months after the accident, roentgenograms were taken to determine the fetal age. In films an oval, calcified ring was noted in the vicinity of the fetal abdomen (Figure 1). In retrospect, this was the mass later discovered within the infant's abdomen at birth, but was interpreted at the time as calcification within a fibromyoma of the mother's uterus.

Upon examination of the infant at birth, a hard, oval mass was noted beneath the abdominal wall just cephalad to the umbilicus. It was approximately 3 by 5 cm. in diameter. Except for undescended testes, no other abnormality was observed.

Roentgenograms taken the day after birth (Figure 2) showed an oval calcified intraabdominal mass 3 by 5 cm. in diameter adjacent to the anterior abdominal wall in the vicinity of the umbilicus. Also visible were a few scattered small areas of calcification elsewhere within the abdomen. Similar calcifications have been reported by Neuhauser[8] as suggestive of meconium peritonitis.

The diagnostic considerations were calcification in a hematoma or abscess or a teratoma. As the infant was asymptomatic, operation was postponed until he was older.

The infant took feedings well and passed normal meconium and subsequently had light yellow stools. He gained weight progressively and remained asymptomatic until November 27, 1949. He was then ten weeks old and weighed 10 pounds, 8 ounces. On that day, he refused feedings and vomited. The abdomen became distended and the baby cried intermittently as though in pain. He was hospitalized with a diagnosis of obstruction of the small bowel. Roentgenograms taken on entry showed no change in the calcified mass. In the next few days the symptoms subsided spontaneously on conservative management.

A week later the patient reentered the hospital with similar symptoms. He was moderately dehydrated and the abdomen was distended and tympan-

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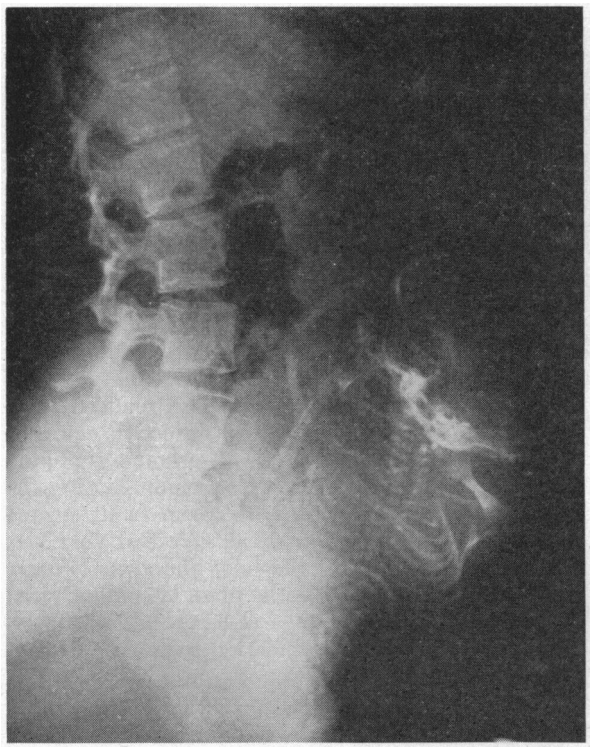

Figure 1.—Roentgenogram of mother's abdomen at eighth month of pregnancy showing calcified ring in fetal abdomen.

itic. The clinical course was that of partial small bowel obstruction with failure to improve, and after hydration he was taken to surgery.

The abdomen was opened through a transverse incision directly over the mass and extending beyond it on both sides, permitting access to the peritoneal cavity. The mass was found to be calcified and about the size of a small chicken egg. It was firmly adherent to the abdominal wall, to the liver and to several loops of jejunum which were kinked and partially obstructed. In dissecting the mass from the bowel, a communication was encountered between its interior and one loop of jejunum through a hole 1 cm. in diameter. The mass was dissected free, the opening into the jejunum was closed and the abdomen was closed in layers. The patient made a good recovery and remained asymptomatic to the time of most recent observation some six years later.

The gross specimen is shown in Figure 3. The wall averaged 0.5 cm. in thickness. Microscopic examination showed it to be composed of dense fibrous tissue with calcified plaques interspersed throughout. There were no endothelial or muscular elements and no structures resembling intestinal wall. Iron pigment was not observed in specially stained sections.

DIFFERENTIAL DIAGNOSIS

There are several diagnostic possibilities in the present case, the most likely of which is a calcified meconium abscess. The others are calcification in a

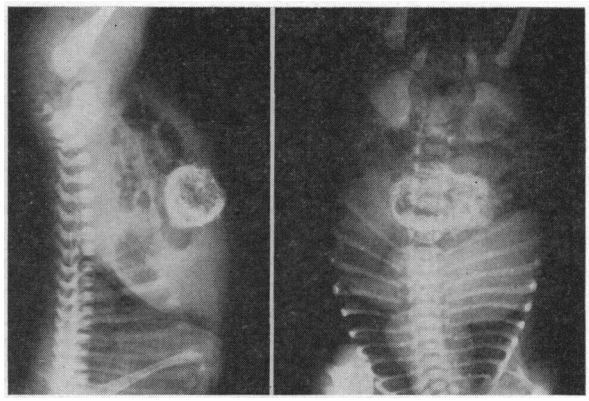

Figure 2.—Roentgenograms of infant's abdomen at birth.

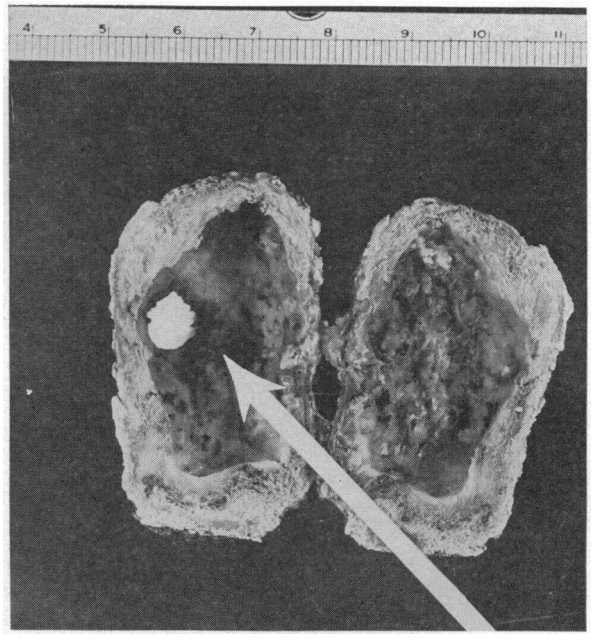

Figure 3.—Photograph of the calcified cyst removed at laparotomy for intestinal obstruction. Arrow points to hole which communicated with jejunal lumen.

hematoma, enterogenous diverticulum, vitelline duct or omphalomesenteric cyst, urachal cyst or mesenteric cyst.

Calcification in a hematoma can almost certainly be excluded by the absence of iron pigment in special stains.

Developmental enterogenous cysts and diverticula and vitelline duct cysts practically always have some demonstrable muscular or epithelial structures resembling small intestine. They rarely undergo calcification although Evans,[5] in a very complete paper on this subject, cited a case of vitelline duct cyst with partial calcification. However, when any connection with the intestine exists in vitelline duct cysts, it is with the ileum. In the case herein reported, the connection was with the upper jejunum.

Urachal cysts are lined with bladder epithelium

and are located caudal to the umbilicus, somewhere between it and the superior surface of the bladder.

Mesenteric cysts occur between the leaves of the mesentery. Vaugn and co-workers[10] reported a case of a calcified mesenteric cyst in an adult with a preoperative appearance by roentgenogram quite similar to that in the present case. However, the location between the leaves of the mesentery established it as a mesenteric cyst.

Calcified Meconium Abscess

By the process of elimination, the most likely diagnosis is a calcified meconium abscess secondary to *in utero* perforation of the jejunum, and meconium peritonitis, sometime prior to the eighth month of fetal life.

In support of this diagnosis was the presence of scattered small areas of calcification elsewhere in the infant's abdomen seen on the initial postnatal roentgenogram. It is well known that sterile meconium in the free peritoneal cavity causes a nonbacterial, foreign body peritonitis and produces dense fibrous adhesions and calcified plaques. According to Boikan,[1] who wrote a very complete paper on the subject, with an interesting discussion of the etiology and pathology, the calcification is most likely due to precipitation of the tissue fluids by the broken-down fatty constituents of meconium. Many case reports of meconium peritonitis mention such calcification.[1,2,3,4,8] According to Litten,[7] it may occur within 24 hours after perforation. Neuhauser[8] described the characteristic roentgenographic picture as almost pathognomonic. It consists of multiple calcified areas diffusely spread throughout the abdomen with occasional larger localized plaques incorporated in the wall of an abscess.

Dubler,[4] as far back as 1888, reported a similar case and he described the pathological observations in great detail. The case was that of a newborn infant who died of pneumonia and at autopsy was observed to have numerous adhesions and calcified plaques and to have a calcified cyst in the abdomen which Dubler considered to be the result of an intrauterine perforation of the descending colon. A translation of his description is as follows: "A cyst the size of a pigeon egg, the wall of which was of irregular contour, nodular and encrusted with chalk. A slide from the wall showed connective tissue and chalky masses but nowhere any hint of a distinct epithelial layer, glandular tubes or muscle fibers." This description very closely fits the calcified, cyst-like structure removed in the present case.

Meconium Peritonitis

Meconium peritonitis is relatively uncommon. Since Simpson[9] reported 25 cases in 1838 there have been a little over 150 cases reported to date. The *Current List of Medical Literature* lists papers reporting about three to four new cases a year.

The etiologic process in cases with primary intestinal obstruction is understandable. Perforation results secondary to the obstruction, which may be due to congenital stenosis, atresia, imperforate anus or meconium stasis. However, 50 per cent of cases occur without obstruction and the explanations of the perforation are not entirely satisfactory. Boikan,[1] after considering the possible causes in detail, concluded that it was due, in the case he reported, to a primary vascular insufficiency of the intestinal wall. Other explanations are trauma during labor or delivery, and congenital and acquired diverticula. Franklin and Hosford[6] encountered two cases in which perforation was found without obstruction at the time of operation. It is conceivable that the abdominal trauma which the mother of the infant, in the case herein reported, received when she was six months pregnant was sufficient to produce an intestinal perforation in the fetus.

The clinical picture of meconium peritonitis is variable, depending on the time of perforation, the amount of spillage and various other factors. The typical picture is that of abdominal distention and severe paralytic ileus secondary to peritonitis, becoming manifest within a few hours after birth. The characteristic calcification on roentgenograms is helpful in determining the etiology preoperatively. The mortality rate in such cases is very high in spite of early intervention. On the other hand, if the perforation has occurred and subsequently sealed in the absence of obstruction, quite some time prior to birth, the resulting peritonitis is sterile and subsides leaving dense adhesions, scattered, small calcified plaques and occasionally a localized encapsulated collection of meconium surrounded by a fibrous wall which becomes impregnated with calcium. The present case is the only one the authors could find on record in which the asymptomatic, calcified cyst was the presenting finding.

The treatment of those infants presenting a picture of acute peritonitis is operative intervention as soon as the diagnosis is made and the condition of the baby permits. It must be realized that time is important, for delay of several hours may mean that a sterile, chemical and foreign body peritonitis will become bacterial. Prompt administration of antibiotics may prevent this. It is wise to empty the stomach by intubation prior to operation but valuable time should not be lost in attempting to intubate and decompress the small bowel. The site of perforation should be identified and closed promptly and the possibility of an underlying obstruction sought and rectified by a bypassing or other suitable procedure if the child's condition permits. Often the latter is not possible and a temporary tube vent or, in the case of a meconium stasis, a double barreled Mikulicz resection and enterostomy may be a life-saving measure.

SUMMARY

A case is presented in which a previously undiagnosed, calcified, intraabdominal cyst produced intestinal obstruction in a ten weeks old infant and was removed at the time of laparotomy for the obstruc-

tion, the infant making an uneventful recovery. The cyst was present and appeared as a calcified ring on roentgenograms during the eighth month of fetal life. The differential diagnosis is discussed and the cyst is concluded to be a calcified meconium abscess. The subject of meconium peritonitis is reviewed.

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Congenital Absence of the Gallbladder

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CONGENITAL ABSENCE of the gallbladder is a rare anomaly of the biliary tract. It is the purpose of this communication to report an additional case and to briefly review the literature. The approximate incidence of this anomaly, derived from autopsy statistics of several reported series, is given in Table 1. It is felt that this is at best an approximation. There are a number of individual case reports that cannot be considered in estimating the incidence, since the total numbers of patients from which these are drawn is unknown. The true incidence can only be derived from autopsy statistics, since absence of a gallbladder shadow in roentgen films, following ingestion of dye, is more likely to be due to a nonfunctioning gallbladder than to absence of the organ.

The overall incidence based on the data in Table 1 is .09 per cent. However, it will be noted that the reported incidence in the pediatric age group (0.37 per cent in data from the Hospital for Sick Children) is considerably higher than the overall incidence. This is probably in keeping with the higher incidence of all congenital anomalies in this age group, since mild deformities which are compatible with life may go unrecognized for many years into adult life, while the more severely incapacitating anomalies usually will be recognized during the early years. Exclusive of the data on the pediatric group, the incidence in the remainder of the combined data in the Table is 0.04 per cent, which is probably more nearly the actual incidence of this anomaly in adults. A more accurate figure could only be obtained by the compilation of a large number of autopsy statistics, since the chance for error with a rare condition and a relatively small sample is quite high.

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The signs and symptoms in patients with congenital absence of the gallbladder who have disease of the common duct are apparently little different from those in patients with a complete biliary tract and disease in either the gallbladder or the common duct, or both. In autopsy material the anomaly is observed about equally often in both sexes. Clinically, however, it is observed about twice as often in females as in males, which accords with the usual sex ratio in surgical disease of the biliary tract.[5] That is, the anomaly apparently occurs as often in men as in women but disease of the common duct requiring operation is twice as frequent in women.[5]

The effect of the absence of the gallbladder on biliary function is not known, as no laboratory studies to determine the effect have been reported. Mouzas and Wilson[8] expressed belief that there is no alteration in function. This opinion is not shared by Caylor[1] who was of the opinion that this anomaly is fairly frequently associated with disease of the remaining intact portions of the biliary tree, especially the common duct. Reports in the literature would seem to bear out this latter opinion. Thus, in a series of 60 cases of congenital absence of the gallbladder reported by Dixon and Lichtman,[2] 58 per cent of the patients had symptoms of cholecystic disease. Of this 58 per cent, 48 per cent had jaundice

TABLE 1.—*Incidence of Congenital Absence of the Gallbladder (Data from Reports of Autopsy Series).*

Data supplied by	No. Cases	No. Autopsies	Incidence (Per Cent)
‡Knox[4]	2	2000	
‡Mentzer[7] and Nagel[9]	1	1600	
‡Lloyd[6]	1	5000	0.04
‡London Hospital*	9	21631	
†Hospital for Sick Children*	20	5395	0.37
Total	33	35626	0.09

*Quoted without reference in Mouzas and Wilson.[8]
‡Presumably series of adult subjects.
†Pediatric data.